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Estimation of Left Ventricular end Diastolic Pressure (Lvedp) from Echo- Doppler Study in Patients of Acute Myocardial Infarction

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ABSTRACT

Background : Heamodynamic consequences of myocardial infarction are expressed as varying levels of LV function. After AMI there is wide variety in the haemodynamic responses .Some patients with small infarct and increased sympathetic tone, LV function may be in normal or in supra normal range. As the size of infarct increases LV function is progressively shifted to down and to the right. The combination of LVEDP and cardiac output is helpfull in assessing LV function. The mean pulmonary capillary wedge pressure provides accurate approximation of LVEDP. When pulmonary vascular resistance is normal , the pulmonary artery diastolic pressure is equal to PCWP (Pulmonary capillary wedge pressure).

Objective : To estimate LVEDP in AMI , irrespective of status of LV function , to prognosticate and as guide for treatment of AMI.

Methods : A hospital based study conducted for two years , included 40 patients who were admitted with AMI in ICCU of BTGH. Patients who had evidence of mitral valve or aortic valve disease or arrhythmias were excluded from study. Detailed information of all patients were recorded as per the proforma any complications if present were also noted.

Result : In this study, AMI is common after 6th decade, male are more prone than females. Important risk factors were smoking, male sex, hypertension, diabetis melitus, family history of CAD. Commonest symptoms of AMI was chest pain with or without sweating other symptoms were vomiting, dyspnea, syncope.

LV Function :37.5% patients had LV diastolic dysfunction, 17.5%had systolic dysfunction,12.5% had combined dysfunction and 32.5% patients had normal LV function.

LVEDP in 1st and 2nd study : many patients had moderate elevation of LVEDP i,e 32 and 24 in 1st and 2nd study respectively, severe elevation in 3 and 6 patient in 1st and 2nd study respectively, mild elevation in 4 and 6 patients in 1st and 2nd study respectively.

Conclusion : LVEDP can be assessed reliably from mitral flow velocity curves in patients with AMI who is in sinus rhythm and who has LV dysfunction. It is possible to predict the prognosis by changes in filling pressures.

KEYWORDS LVEDP, AMI, coronary artery disease, Ecocardiography, PCWP (pulmonary capillary wedge pressure.) E velcity , A velocity, E/A, DT, AFF, IVRT,

Introuduction

Haemodynamic consequences of myocardial infarction are expressed as varying levels of LV function. After AMI there is wide variety in the haemodynamic responses .Some patients with small infarct with increased sympathetic tone, LV function may be normal or supra normal range. As size of infarct increases LV function is progressively shifted to down and to right.

The combination of LVEDP and cardiac output is helpful in assessing LV function. The mean pulmonary capillary wedge pressure provides a reasonably accurate approximation of LVEDP. When pulmonary vascular resistance is normal , the pulmonary artery diastolic pressure is equal to PCWP (pulmonary capillary wedge pressure.)

The prognosis and clinical status in AMI are related to both the cardiac output and PCWP (LVEDP).[1,2] It is possible to measure both parameters by invasive method by inserting swanganz catheter. Patients with normal cardiac output after AMI have extremely low mortality. Prognosis worsens as cardiac output declines. Patients with cardiac index in range of 2.7 to 4.3 liters/min/m^2 usually have no signs of impaired perfusion. Patients with cardiac index in range of 1.8to 2liters/min/m^2 usually show early signs of hypoperfusion. Patients with cardiac index less than 1.8liters/min/m^2 are usually in shock.

LVEDP by ECHO doppler study.[3]

LVEDP is influenced by both systolic as well as diastolic function. Conventionally cardiac haemodynamic data have been obtained by invasive cathetarization techniques. Now it is possible to determine various haemodynamic parameters noninvasively using Echocardiography. M mode and 2D echocardiography provide only indirect evidence of haemodynamic abnormalities, but this evidence may be initial clue. Intracardiac haemodynamic assessment is best performed by doppler echocardiography. The accuracy of doppler derived haemodynamic measurments has been validated and confirmed by simultaneously derived catherization data.

Since left atrial pressure is one of most important determinant of diastolic filling profile, it can be estimated from various diastolic filling velocity varibles.[4]

Mitral flow velocities are measured by pulsed wave doppler with sample volume placed between the leaflet tips and the following diastolic filling parameters are derived.

IVRT : Isovolumic relaxation time. (2) E : early filling velocity.

(3) A : Late filling velocity. (4) Dt of E : deceleration time of E.

(5)AFF : atrial filling fraction, TVI of A / TVI of total mitral inflow.

(6) Dt of A : deceleration time of A.

(7) TVI of E : time velocity integral of E

(8)TVI of A : time velocity integral of A.

(9) E/A : time velocity integral of E / time velocity integral of A.

(10)MAR : is interval from end of A wave to R wave on ECG.

The formula used to calculate LVEDP is as follows

LVEDP = 46 – 0.22 IVRT – 0.10 AFF – 0.03 DT – (2 / E/A) + 0.05 MAR.

LVEDP may increase with systolic dysfunction or diastolic dysfunction or both systolic and diastolic. So it is important first to know about assessment of LV dysfunction by 2 D echocardiographic examination.[5]

Materials and Methods.
A hospital based study was conducted for 2 years involving 40 patients admitted with AMI in BTGH. Informed consent was obtained, Patients who had evidence of mitral valve disease or aortic valve disease or arrhythmias were excluded from study. The diagnosis of AMI was done on typical history, ECG changes, and serum enzymes. A detail information of all patients were recorded as per the proforma, any complication if present were also noted. Drugs used for treatment of patient during study period were documented. Investigation like ECG, CPKMB, Echo colour doppler study were done in all patients.

Echo colour dopplet study. Machine used : ATL ultrmark 6 with colour doppler facility. Systolic function was determined by M mode echocardiographic measurement by using apical 2 chamber view. Wall motion abnormality, any complication like pericardial effusion, thrombus in left ventricle , MR, VSD etc noted.

Measurment of LVEDP : Mitral flow velocities measured by pulsed wave doppler with sample volume placed between leaflet tips. Various parameters used as following…

(1) Peak rapid filling velocity (E peak M/sec) (2) A wave : (A peak m / sec)

(3)TVI of E : is area under E wave or the time velocity integral reflects the contribution of rapid filling phase of left ventricular diastolic filling.

(4) TVI of A : time velocity integral of A wave is the area under A wave and reflects the contribution of atrial contraction to left ventricular diastolic filling.

(5) DT of A : (Msec) : it is represented by the time interval between the A peak and point on baseline intercepted by deceleration wave form.

(6) DT of E : (Msec) It is represented by the time interval between E peak and the point on baseline intercepted by deceleration wave form.

(7) IVRT : (isovolumic relaxation time) measured by positioning sample volume of pw doppler halfway between anterior mitral leaflet and left ventricular outflow tract. The time between the end of the left ventricular outflow velocity waveform and onset of mitral inflow velocity wave form represents IRP (isovolumic relaxation period).

(8) TVI E / TVI A : Time velocity integral of E / Time velocity integral of A.

(9) TVI on MI (TVI of mitral inflow) is the total integral of mitral velocity.

(10) AFF (Atrial filling fraction) is fraction if inflow volume resulting from atrial contraction.

AFF = TVI of A / TVI of MI.

(11) MAR : is time interval between termination of mitral inflow (i.e end of the A wave) and the R wave on ECG. This interval was expressed as +ve if mitral velocity ended before the R wave and – ve if the velocity ended after R wave on ECG.

The formula used for calculating LVEDP is as follows,

LVEDP = 46 – 0.22 IVTR – 0.10 AFF – 0.03 DT – (2 – E/A) + 0.05 MAR

Results.
MI is common in age 60 years and above, males are more prone for MI. Chest pain was most common symptom seen in 90% patients in our study , other symptoms were sweating, vomiting, dyspnea, nausea , syncope. Important risk factors were smoking (65% patients) , Hypertension (12.5% patients) , Diabetis melitus (12.5% patients) , family history of coronary artery disease (2.5% patients).

LV function : It is observed from table no 1 that 7 patients out of 40 patients had systolic dysfunction(17.5%), 15 had out of 40 had diastolic dysfunction(37.5%), and 5 out of 40 patients had combined dysfunction(12.5%), and 13 out of 40 had normal LV function (32.5%).

Type of MI : 80% patient had Q wave MI (32 out of 40 patients) , and 20% patient had Non Qwave MI (8 out of 40 patients). Table 2.

Wall motion abnormality : LV wall motion abnormality seen in only in Q wave MI patients, wall motion was normal in non Q wave MI patients.

LVEDP in 1st and 2nd study.
As seen in table 3, LVEDP was normal in 1 and 4 patient is 1st and 2nd study respectively , Mild elevation of LVEDP seen in 4 and 6 patients in 1st and 2nd study respectively , moderate elevation of LVEDP seen in 32 and 24 patients in 1st and 2nd study respectively, Severe elevation of LVEDP seen in 3 and 6 patients in 1st and 2nd study respectively.

LVEDP and Ejection Fraction in 1st study.
As seen in table 4A , 28 patients out of 40 (70% pts) had normal EF i,e more than 50%, among these 28 patients 1 patient had LVEDP of less than 15 mmhg. Mild elevation (15 – 20mmhg) seen in 6 patients , moderate elevation (21 – 30 mmhg) seen in 19 patients , Severe elevation of LVEDP (more than 30mmhg) seen in 2 patients.

Patients EF between 40% - 49% were 7 in nos (17.5%) out of these 1 pt had mild elevation LVEDP , and 6 pts had moderate elevation of LVEDP.

Patients with EF of 30% - 39% were 4 in nos (10%), all these patients had moderate elevation of LVEDP,(21 – 30 mmHg).

Patients with EF between 20% - 29% was only 1 in no who had severe elevation of LVEDP.

LVEDP and ejection fraction in 2nd study.
As seen in table 4B, patients with EF more than 50% were 27 in nos, out of these 27 patients 4 patients had normal LVEDP , 5 patients had mild elevation of LVEDP, 13 patients had moderate elevation of LVEDP , 5 patients had severe elevation of LVEDP.

Patients with EF between 40% - 49% were 9 in nos. , 8 out of 9 patients had moderate elevation of LVEDP, and 1 had severe elevation of LVEDP.

Patients with EF of 30% - 39% were 3 in nos. , all 3 patients had moderate elevation of LVEDP.

Patients with EF of 20% -29% was only 1 in no, who had mild elevation of LVEDP.

Correlation between LVEDP and various parameters of transmitral flow.
(1) LVEDP and IVRT : r = - 0.898 , in 1st study and r= - 0.76 in 2nd study. This shows that co – rrelation between LVEDP and IVRT is highly significant and shows Negative correlation.

(2) LVEDP and DT : r = - 0.35 in 1st study and r = -0.31 in 2nd study. This shows that LVEDP and DT has significant Negative correlation.

(3) LVEDP and E/A : r = 0.36 in 1st study and r = o.48 in 2nd study. This shows that LVEDP and E/A has Highly Positive correlation.

(4) LVEDP and AFF : r = - 0.26 in 1st study and r = - 0.21. This shows that there is Insignificant correlation between LVEDP and AFF.

(5) LVEDP and MAR : r = - 0.26 in 1st study and r = 0.008 in 2nd study. This shows that there is no correlation between LVEDP and MAR.

Discussion.

The two major determinants of LV filling are …

(1) Ventricular relaxation.

(2) Effective chamber compliance.

Ventricular relaxation is complex energy dependent process during which the cotractile elements are deactivated and the myofibrils return to their precontraction length.[6] In normal heart , ventricular relaxation begins during midsystole and continues throughout the first third of diastolic filling. In the cathetarization laboratory, relaxation abnormalities are measured from LV pressure obtained with high fidelity manometer tipped catheters. Peak negative change in LV pressure over time (dt / dt) and the time constant of relaxation , are accepted indices of rate of relaxation, although both have limitations. In disease state, relaxation abnormalities occur early, often preceeding dysfunction of contraction phase[7].

The effective operating chamber compliance describes the passive properties of the LV during blood flow across the mitral valve from LA to LV. Several complex interactions occur during this period , including the continued effect of ventricular relaxation , diastolic suction, passive filling, pericardial restraint, ventricular interaction and visco – elastic forces of the myocardium.

In 1982 Kitabatake et al described, the transmitral flow velocity curves obtained with doppler echocardigraphy in different disease states.These mitral flow velicity curves correlates well with the first derivative of diastolic volumetric flow rates obtained by other accepted methods such as left ventriculography, radionuclide angiography and digitised M-mode echocardiography. It is now possible to predict prognosis , estimate filling pressures and guide the therapy by analysing mitral flow .[8,9,10]

The doppler determinants of mean LA presure occur in early diastole and are measured by the E velocity and deceleration time. The initial E velocity is influenced mainly by driving pressure from the LA to LV at the time mitral valve opening and deceleration time is determined by effective operative compliance of the left ventricle. Thus in many patients with a high mean LA pressure, E velocity is usually high and deceleration time is short, on the transmitral flow velocity curve. Conversely, if the LA pressure is low, the initial E velocity is usually low and the deceleration time is prolonged. Several studies of patients with severe LV systolic dysfunction including our present study, the inverse correlation od Dt with LVEDP is excellent. Short deceleration time of less than 150ms, nearly always indicates a LA mean pressure more than 25mmHg. Conversely a prolonged deceleration time correlated with LVEDP of less than 15 mmHg. A high E/A ratio (more than 2.5) is associated with high LVEDP and low E/A ratio is associated with low LVEDP in patients with LV systolic dysfunction.[11,12,13] Normal subjects abnormality of diastolic filling but because of rapid ralaxation and rapid suction effect. The correlation between DT and LVEDP becomes poorer, which makes it difficult to determine LVEDP in specific patients on the basis of mitral flow velocity curve alone. Now technique are being investigated for measurement of LV filling pressure viz pulmonary vein velocity curves, colour M- mode echocardiography[14-17] which measures the flow propagation into LV and Doppler tissue imaging which directly examines myocardial motion.

Summary and Conclussion.

LVEDP can be assessed reliably from the mitral flow velocity curves in patients in sinus rhythm who have LV systolic dysfunction. It is possible to predict the prognosis by analyzing mitral flow. The estimate of LVEDP provided by the equation could be used to track changes in filling pressures after an intervention. **The pointers of elevated LVEDP are IVRT of < than 60msec, DT < 130 msec, E/A > 2.0 , AFF < 20%.**

All patients except one showed elevated LVEDP in first study. In subsequent study in only 3 patients LVEDP returned to normal. LVEDP of more than 30mmHg was noted only in patients with AMI who had LVEF > 40%.This could be because of diastolic dysfunction alone. Negative correlation between LVEDP and IVRT is Highly Significant.

Highly significant positive correlation was noted with LVEDP and E/A ratio.

LVEDP and DT showed limited negative correlation.

Table no 1 : LV function.

	Systolic dysfunction	Diastolic dysfunction	Combined dysfunction	Normal function
No of patients	7	15	5	13
%	17.5%	37.5%	12.5%	32.5%

Table no 2 : Type of MI

S.N	Type of MI	No of patients	%
1	Q wave MI	32	80%
2	Non Q wave MI	8	20%

Table 3 : LVEDP in 1st and 2nd study.

SN	LVEDP	No of pts in 1st study	No of pts in 2nd study
1	Normal LVEDP (<15mmHg)	1	4
2	Mild elevation (15 to 20mmHg)	4	6
3	Moderate elevation (21 to 30mmHg)	32	24
4	Severe elevation (>30mmHg)	3	6
	Total	40	40

Table no 4A : LVEDP and ejection fraction (EF) in 1st study.

SN	EF	Normal LVEDP <15	Mild elevation of LVEDP (15 – 20)	Moderate elevation of LVEDP (21 -30)	Severe elevation of LVEDP >30	Total
1	Normal >50% (28pts)	1	6	19	2	28
2	40% -49% (7 pts)	–	1	6	–	7
3	30% - 39% (4 pts)	–	–	4	–	4
4	20% - 29% (1 pt)	–	–	–	1	1
Total No of Patients	40					40

Table No 4B : LVEDP and LVEF 2nd study.

SN	LVEF %	LVEDP normal < 15	Mild elevation 15-20	Moderate elevation 21 - 30	Severe elevation > 30	Total
1	Normal >50	4	5	13	5	27
2	40 - 49	–	–	8	1	9
3	30 - 39	–	–	3	–	3
4	20 - 29	–	1	–	–	1
					Total	40

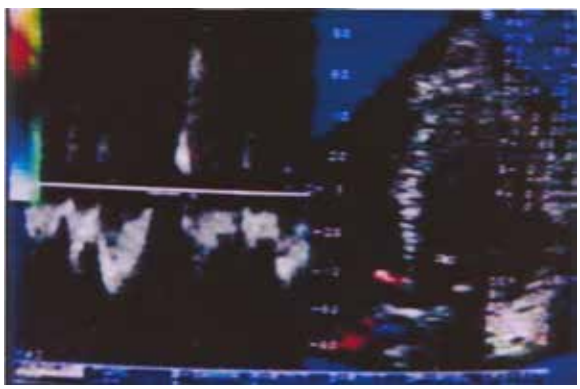

Figure 1: Measurement of IVRT

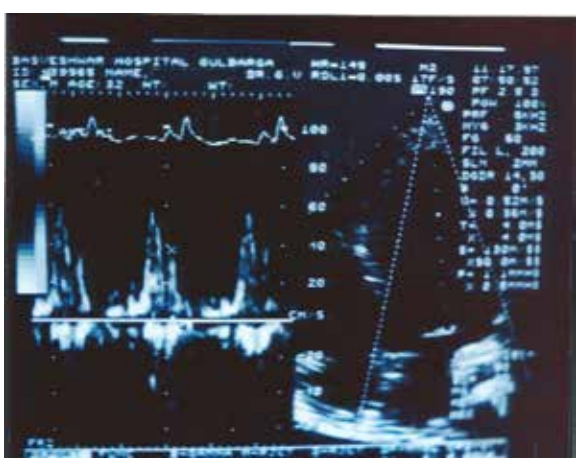

Figure 2: Maximum LVEDP Noted (33.8mmHg)

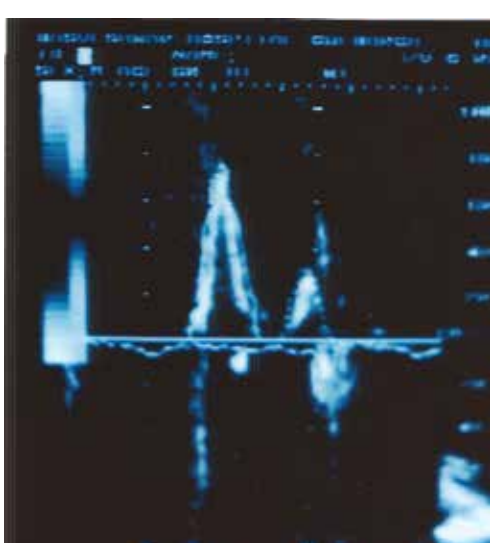

Figure 3: 1st Study Showing Restrictive Pattern

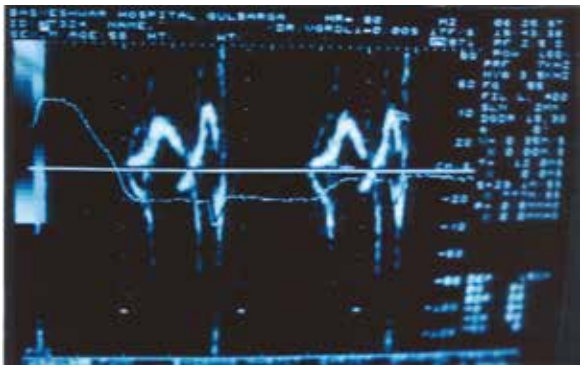

Figure 4: 2nd Study Showing Abnormal Relaxation

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